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ABSTRACT

Emphasizing that for any known teratogen no safe dosage level exists, this case-illustrated review identifies the bases for current concern about the pollution of the environment, reflects on the promise and complexities of the emerging disciplines of behavioral toxicology and behavioral teratology, and describes existing evidence of teratogenic effects of toxic chemicals. Bases of concern include (1) the need for bureaucracies to create environmental limits to toxic concentrations; (2) the tendency for the burden of proof of toxic effect to lie with the afflicted; (3) the concept of a safe dose level; and (4) the influence of politico-economic considerations on decisions to improve environmental control of effluents and industrial emissions. Discussion of the currently available evidence of toxic effects focuses on polychlorinated biphenyls, diethylstilboestrol, vinyl chloride monomer, manganese, mercury, and, extensively, lead. Recommended actions to be taken to adequately protect citizens from chemical assault are offered, and the paper concludes with an account of evidence of nuclear take-up by children living near British nuclear installations and the lack of adequate official response to such evidence. (RH)

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THE VIOLATION OF CHILDHOOD: A REVIEW OF POSSIBLE EFFECTS ON DEVELOPMENT OF TOXIC CHEMICAL AND NUCLEAR WASTE

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In January 1985 an article appeared in the British periodical 'New Society' which drew attention to inexplicable cases of microphthalmia and anopthalmia in children born to families living in the vicinity of two incinerating plants. Both plants were operated by the same company and both existed commercially to incinerate highly toxic chemical and industrial waste. The teratogenic effects were also evident in farm livestock, notably calves, born in the vicinity of both plants and in some domestic animals. The pattern of birth defects could be a coincidence; there is no proof, as New Society was at pains to point out, that the emission of combustion products of polychlorinated biphenyls into the atmosphere, or the combustion product of any other material for that matter, is associated with the observed birth abnormalities. The facts, such as they exist, would appear to be as follows; the two plants are separated by a distance of over 350 miles, one is in Wales, the other in Scotland; a potent carcinogen and known teratogen 2378TCDD, a dioxin, has been detected in the soil near both plants; 2378TCDD is a possible product of the incomplete compbustion of polychlorinated biphenyls; near the Welsh plants, four children with rare eye defects were known to have been conceived within four years - the figure in Scotland was higher at five children within a thirteen month period. Between them, these nine babies exhibit a range of abnormalities from complete absence of eyes to absence of eyelids; some have just one eye.

There is little medical knowledge as to what causes the defects but expert opinion suggests that one such case a year from a total population of 10 million would be an appropriate order of magnitude. In the case of Wales, such a population size is roughly three times the actual national total population. To put this in perspective, given the postulated incidence of disorders of the kind described, then throughout the entire country one would not expect to observe one such case per year. The fact however, is that within a ten mile radius of one small Welsh town, four such cases have been found in four years, and within that same area local cattle have been born blind, or with one eye.

The Scottish context is essentially similar. Dioxins are evident in the soil of both areas and are entering food chains. In both areas there are other industrial processes intrinsically capable of generating dioxin contaminated effluent and releasing these into the environment - municipal incineration near the Scottish plant and numerous industrial smoke stacks near the Welsh plant.

Toxic material where it is released as industrial emission in airborne form finds its way into the human blood stream through a number of routes and influences current and future generations in a manner that is most surely complex. Contingent issues relate to age and stage of development, general state of health, social factors of all kinds, level of dosage received from all sources at a particular time, the proportion of the total ingested toxin that is absorbed by different routes, the duration of exposure, even to low level contamination and the tendancy of the toxin to destroy or modify genetic material. An important associated factor in so far as it is significantly related to procedures for environmental control, is manifest in the recognition or otherwise of safe-dose levels for pollutants of all kinds. Such limits, where

they are set, are based on the premise that there is a level of toxic concentration in human blood and tissue below which intellectual functioning, motor performance and behaviour, neurological functioning and the normal bodily processes of growth and repair are all uninfluenced by the invading material. Current concern, as it is expressed in this Paper grows out of the following:

1. The need for bureaucracies to create environmental limits to toxic concentrations in for example, water suplies, air, foodstuffs, soils, public buildings through assumptions related to the relative contributions to total body burdens of toxic compounds from identified sources and further assumptions relating to the differential absorption of such compounds in e.g. the gut, the lower respiratory tract etc.

2. The tendency, particularly in the U.K., but not limited to these islands, for the burden of proof of effect to lie with the afflicted. Those who have been damaged, frequently do not have the resources to prove their case in protracted expensive legal battles. A more humane and responsible view would require industrial polluters to prove that they have not and will not cause damage by their operations.

3. The concept of a safe dose level, per se. To what extent is such a notion consistant with what is known about the variability of behavioural response to environmental stimuli? All representations in respect of safe dosage emerge from the evidence gained as a result of clinical, experimental and epidemiological enquiries, including animal and human volunteer studies. Reviews of such evidence, notably in the case of environmental lead, can not provide **evidence of lack of effect** in low dose subjects because of a variety of confounding factors, often social in origin, (see e.g. Smith, 1985, Rutter 1980) criticisms of experimental design and control (Rutter 1980) and insufficiently sensitive criterion-performance measures. (Gregory & Mohan, 1977). I have deliberately inverted the general conclusions of the studies cited which are frequently apt to state their conclusions in terms of no conclusive evidence of effect. Given that toxic substances by definition have an affect on man, and neuro-toxins in particular attack the central nervous system, my personal philosophy is to subscribe to the view that 'no evidence of effect may be the effect of no evidence' with the implications that this carries for the public control of pollution.

4. The improvement of environmental control of effluents and industrial emissions is closely allied to politico-economic considerations. It is rare that pollution and toxic emission cannot be reduced; it is more usual that public health and in particular the future health and development of the infant and young child is consciously put at risk. The concept of the safe dose level seeks to minimise the risk but by definition clearly does not seek to erradicate it. It is within the 'minimal-risk' concept that the danger for young children resides. The nature of the human organism is such that it is capable of idiosyncratic response to ecological variation and disturbancy of body chemistry. Extremely rare occurances are 'lost' within risk calculations since the statistical methods underpinning such actuarial calculations are ill-equipped to deal with a few isolated

cases. By the same token statistical procedures employing population base rates for various childhood disorders are frequently employed by public corporations and governments to explain away a postulated link between child health and a local environmental hazard. I shall return later in this Paper to precisely this point in relation to radioactive emission in the vicinity of nuclear facilities, particularly in the UK.It should be apparent that the search for proof of a causal link between an environmental factor and a low occurance human deviation or pathological growth response is appropriately dealt with by accumulating the circumstantial or coincidental evidence rather than by dismissing the evidence of coincidence as anti-scientific. An example may illustrate my point.

Until fairly recently the notion of a link between childhood behaviour and diet would have received little credence from the scientific community. Feingold's (1973, 1975) early assertions on a link between behavioural disorder such as hyperkinesis and various environmental chemicals and food additives has created dispute and debate. Nevertheless, Weiss (1982) in an influential review of controlled studies argues that "the Feingold hypothesis, in principle, is supported by experiments that meet scientific criteria of validity". Weiss (op cit) also goes on to note that the developing discipline of behavioural toxicology (Weiss and Laties 1975)has evolved in response to the recognition that 'adverse effects of environmental agents need not be limited to tissue pathology". Effects on behaviour can be just as profound and disabling. The environment contains many neuro-toxins. I have already mentioned the issue of dioxins; other environmental factors include radio elements, heavy metals, e.g. Pb, industrial gases such as SO2, CO and pesticides. The effects of all of these on the foetus and neonate are of immense concern and it is the methodologies of behavioural toxicology and behavioural teratology in particular that are likely to be illuminating in the future.

In the above example there are a number of points worth emphasising:

Firstly, the Feingold hypothesis constitutes the postulation of a link based on observation and clinical evidence.

Secondly, the postulation of a link is explored by a logical set of experiments which seek to monitor behavioural change induced by a diet free of the postulated toxics. On the basis of the notion of idosyncratic response, the particular food addititves will of course vary from child to child, and the methodology includes attempts to explore this.

Thirdly, the statistical protocols are necessarily sensitive to the postulated idiosyncratic nature of response to therapy.

Fourthly, there is a clear recognition that whilst dose response information is important from a scientific viewpoint, the whole field is dogged by a virtual absence of such information.

Let me return briefly to the issue of environmental contamination by 2378TCDD at Pontypool in Wales and Bonnybridge in Scotland. This chemical is one of the most potent known carcinogens; in concentrations of 1 part per billion it has been shown to have a teratogenic effect on mice. Its dose response levels in humans, particularly in the developing foetus, is completely unknown. Although the levels of this dioxin found near Pontypool are reported to 1.1 parts per trillion, expert opinion on chemical toxicology (see Sweeney 1985) suggests that the total dioxin concentration can properly be regarded as 200 times greater than the noted concentration of 2378TCDD. This could bring the concentration dangerously close to the levels known to cause teratogenic effects in mice. This however simply detracts from the main point, namely that the most sensible position to take is that there is no safe dose. Equally, the view that there is a background level in the environment which is 'safe' is spurious. These are man made toxins; if they are in the environment then man has put them there. It is of course quite the case that within the 'natural' environment man's phylogenetic pattern of adaption has, over time, been set against background levels of potential neuro-toxins such as lead and manganese as well as irradiation from cosmic and terrestrial sources. Within Roman civilisation the overt symptoms of clinical lead poisoning were by no means unknown. Industrialised societies have a vast appetite for mineral resources of all kinds, the requirement for new synthetic materials to match the demand of new technologies, industries and markets has significantly altered the chemical environment to which man is exposed and elevated the 'background' level of significant neurotoxins. "Nowhere on the earth's surface, with the exception of some remote ocean depths, can now be regarded as unpolluted. No human populations, however, remote, can now be regarded as unexposed". (Bryce-Smith & Stephens, 1980). Figures from the U.S. National Academy of Sciences (1980) suggest that the present day lead level in the urban environment is up to 10,000 times greater than estimated 'natural' levels; even in remote rural areas the ratio of present day/natural levels can be as high as 1000:1 and food lead levels up to 100 times greater than their 'natural' level. The Lead in Food Regulations, U.K. (1979) stipulate a maximum lead level of 1 part per million in most fresh food, and a maximum of 0.2 p.p.m. in food for infants. The Conservation Society (1980) point out that because of the airborne nature of much lead contamination and the affinity of lead for leafy vegetables, many food crops grown in urban gardens will be statutorily unfit for human consumption. In fact the same report urges that home grown food in urban areas should not be eaten by pregnant women, infants and young children. Rom (1976) has pointed out that for nearly 100 years high lead exposure has been associated with abortion and stillbirth in both occupationally exposed women and in the wives of male lead workers. The effects of lower lead body burdens on pregnant mothers and developing children is a matter to which I shall return.

Of particular note in the case of lead is the politico-economic climate which arguably is responsible for maintaining environmental lead concentration at their present levels through failing to enact legislation banning lead derivatives in petrol. This is not the case in some other European countries nor in North America where ten years or more of monitoring have illustrated the fall in blood lead levels in

children as a result of preventative policies which include the prohibition of lead additives in petrol. The way in which lead and other metals in the environment may affect children I shall review briefly in the next section. On the more general point of contamination of the modern urban and rural environment, both directly and indirectly,Barlow (1980) also draws the important distinction between exposure to environmental agents, drugs or industrial chemicals preconceptually and post-conceptually. It is argued that "exposure of males or females at any time may cause abnormalities of the germ cells which, if they do not result in parental infertility, may cause late emerging problems for a subsequent conceptus".

The point is an important one, since the conceptus itself does not necessarily have to be exposed directly to environmental agents for the effects of parental exposure to manifest later in the post natal development of the child. Behavioural teratology is essentially concerned with late emerging effects and this has been touched on earlier. Some environmental agents do, of course, result in quite obvious and severe birth defects - thalidomide is a particularly tragic instance. Effects produced by other agents are delayed in time and their manifestation frequently complicated by 'normal' socialisation and growth processes. By middle childhood and adolescence the possibility of separating behavioural teratological effects from 'social' effects becomes extraordinarily difficult. The more so since parental exposure may not be identified as such at the time it occurs, or, the active agent is not known to be present in the environment or a known chemcial agent is not known to be teratogenic.

Sharratt (1980) elaborates this point and emphasises that we are really in the first decade of inquiry into techniques for investigating the mutagenic activity of chemicals and into determination of mutagenic potential of a wide range of chemical materials. Sharratt (op cit) argues that the term 'mutagen' can be confusing for two reasons: firstly it is unlikely that the majority of chemical damage to genetic material is inherited; secondly many of the tests devised to demonstrate chemical propensity for genetic damage have 'an hereditable mutagenic change in a test organism' as their end point. In consequence the term 'mutagenic chemical' has increasingly been used to refer to materials which react with genetic material in such a way that it is modified or damaged. His analysis of genetic mechanisms suggests that a chemical or its mutagenic metabolite may pass from an exposed mother to the foetus, inducing either abortion or cancer or "if exposure occurs during the period of organogenesis, producing congenital abormalities by a teratogenic rather than a mutagenic mechanism". These mechanisms are for the most part theoretical and considerable epidemiological and clinical difficulties exist in demonstrating mutational changes as a consequence of exposure. Toxicological assessment of chemical agents, which would include its mutagenic activity are abnoramlly difficult given that health risks are "potential" rather than real. (Sharratt op cit).

The observations of Barlow cited earlier in relation to teratogenesis are developed in a discussion of 'Safety at work for Embryos' in a paper by Renwick (1980). The theoretical occurance repeatability of teratogenic effects is emphasised in contradistinction to the random

attack of DNA by a mutagen. Renwick suggests that the problem for occupational medicine is the interpretation of the observation that repeatability is lower than expected, creating e.g. an abnormality in only one embryo of a monozygotic twin pair. "The variation is perhaps best thoguht of as reflecting the importance of the precise timing of the insult. An embryonic tissue may at one moment be just ripe for damage and a few hours later it may be resistant". (Renwick, op cit). The paper also offers a practical or working definition of a teratogenic dose level as one that "kills some cells but not sufficient cells to kill the whole organism; or one that deranges development in a way that is not immediately lethal".

The vulnerability of the embryo to circulating toxins is certainly greater than that of the mother and the placenta offers little barrier to assault even for heavy metals such as Mercury and Lead. This is problematic for industrial toxicology and occupational medicine since a teratogenic dose for the embryo may be unassociated with clinical symptoms of poisoning in the mother or with blood concentration levels which may be within current safe dose limits. It is also the case for industrial workers that the pattern of exposure to substances is seldom known with any accuracy and "analytical systems are usually organised to control exposure below some set limit rather than determine actual exposure" (Sharratt 1980) A point made earlier.

It is the nature of the current situation that teratology is as yet a poorly developed science. Barlow (1980) argues that animal experimentation has enabled the formulation of some general principles - "... Some teratogenic agents given during embryogenesis, at doses below those causing gross structural malformations, may result in post-natal abnormalities of development and behavior; some agents given after the time of embryogenesis but whilst brain development is still in progress may also produce postnatal changes, and that some agents which are not gross structual teratogens may nevertheless be behavioural teratogens."

It is important to note that the term agent may apply to a chemical from any environmental source, whether ingested voluntarily by prescription or advice, or involuntarily through its presence in air, water, food and dust. In the case of drugs, the U.K. Committee on Safety of Medicines introduced a requirement for reproductive toxicity testing of drugs in animals (Barlow 1980), and such studies have proved illuminating (see e.g. Davis et al 1973, Lodge et al 1975, Hill et al 1974 and Koos and Longo 1976). In the U.S.A. the Toxic Substances Control Act of 1976 which requires toxicity evaluation of all new industrial chemicals requires them to be subjected to behavioural teratological or toxicological criteria. Weiss (1982) points out however that in spite of the experimental support for the Feingold hypothesis there is still an absence of behavioural criteria from food additive test protocols.

Not all workers agree on the value of animal studies in the quantitative extrapolation of teratogenic effects. Renwick (op cit) regards such studies as virtually valueless in the assessment of human risk and argues that data from exposed human populations have to be the only basis for quantitative assessment.

WHAT IS THE NATURE OF SUCH EVIDENCE?

1. Polychlorinated biphenyls (PCB) are widely used in industry. The combustion of these compounds under incorrect conditions can, as I mentioned earlier, produce carcinogenic and teratogenic effects. Directly however, PCBs have been responsible for devastating human consequences through industrial accidents. In 1968 consumption of PCB-contaminated cooking oil gave rise to 1300 case of chloracne in adults and cases of small for dates babies with abnormal cola-coloured skin and eye discharge. Kuratsune et al (1972) report that some affected babies were born to symptomless mothers. Later effects to the babies exposed 'in utero' included gingival hyperplasia. Follow-up of a small group of children who ingested PCBs through breast milk only, revealed hypotonia and apathy. **The effects of direct occupational exposure to low levels of PCBs are unknown.**

2. Bibbo et al (1977) and Herbst et al (1974) have invstigated transplacental carcinogenesis by clinical observations on female and male offspring of women given diethylstilboestrol (DES) during pregnancy for threatened abortion. Pre-natal DES exposure, has, on some evidence resulted in a rare form of vaginal cancer which presents around the time of puberty. The incidence of this form of cancer does not appear to be dose related.

3. Maltoni and Lefemine (1974) report that exposure of pregnant rats to vinyl chloride monomer (VCM) can cause angiosarcomas of subcutaneous tissue in the offspring. The long term incidence of cancer in the children of VCM workers remains to be evaluated. AS Barlow (1980) points out, investigations of industrial chemicals has been rare.

4. Clinical signs of Manganese poisoning were initially observed by Couper (1837) in miners handling MnO2 minerals. Frank symptoms included hypokinesia, akinesia, rigidity and tremor and mask like faces. Not suprisingly perhaps the highest incidence of Manganese poisoning occurs in the mining communities of Chile, India, North Africa, USSR and Cuba. Mena (1980) reports that in India up to 25% of the exposed work force of between 30,000 and 100,000 show symptons of poisoning. Kawamura (1940) reported on Manganese poisoning in the general population as a consequence of ingesting Manganese from contaminated drinking water. Tanaka 1969 and Cook et al 1974 report clinical evidence of Mn poisoning associated with ore-crushing plants and steel foundaries in the U.S.A. Based on symptons identified in Chile, the course of intoxification may proceed through psychotic behaviours, including hallucinations, delusions and compulsions and lead into neurological symptoms with associated rigidity, slowness of movement, impaired speech and diminution of postural reflex. Neff and Cosata 1969 indicate the similarity between Manganese poisoning and degenerative diseases e.g. Parkinsonism, in which structural damage to the brain is a feature. A possible factor affecting susceptibility has been linked to increased rates of intestinal absorption of Fe and Manganese in individuals showing iron deficiency anaemia. Mena et al (1978) argue that it is crucial to consider the implications for mother and foetus during pregnancy, given that the intestinal barrier to manganese is not fully developed even in the new born, more particualrly if the birth is

premature. The risk of manganese intoxification of the foetus or infant is high if the mother is exposed. Mena (1980) suggests that recent studies of premature infants have shown markedly increased intestinal absorption of manganese.

5. The possible effects of environmental mercury are reasonably well known and organic mercury compounds are potentially devastating in their impact on both adults and unborn children. Barlow (1980) argues that Methyl Mercury is firmly established as a behavioural teratogen in man. The potential damage that mercury might do to the developing central nervous system of the human embryo is well documented in the Minamata Bay exposure (Harriss and Hohenemser 1978). In the 1950s mercury discharged from a plastics factory into Minamata Bay entered the food chain of local residents via fish caught in Bay waters and substantially in the form of Methyl Mercury. In the period 1955 - 1959 up to 6% of all children born in the area suffered some degree of cerebral palsy and by 1971 in utero exposure was thought to be directly responsible for 25 cases of severe brain damage. A further incident of organic mercury poisoning occurred in Iraq during 1971 due to the human consumption of seed grain dressed with an anti-fungicide of organo-mercury origin. Apart from direct exposure through consumption of the grain as bread, the compound entered food chains in a complex manner via rats and birds. This particular incident generated new international controls for seed dressings by the World Health Organisation.

In the Irag incident the clinical picture of poisoning was dominated by CNS involvement,visual defects and cerebellor ataxia. A significant feature of the incidents in both Iraq and Japan were observations that foetal Methyl Mercury poisoning was evident in cases where mothers often showed no clinical signs of poisoning themselves. (Harada 1968, Amin-Zaki et al 1974).

The critical organ following exposure to Mercury varies with the type of compound, its dose level, route of absorption and the stage of development of the organism. Absorption of Mercury through the skin cannot be ignored and this may be important in the case of airborne Mercury. Its presence in the environment varies enormously but the human contribution to the total annual release is thought to be of the order of 20,000 tonnes. According to Waldron (1980) in the top 30 cm of mud in San Francisco bay the mercury level has been found to vary from 20 p.p.b. to 2,000 p.p.b. with a maximum value of 6430 p.p.b. (p.p.b. = parts per billion). In the bottom sediments of Minamata Bay concentrations were as high as 2,000,000 p.p.b. Down stream of a Mercury cell choralkali plant in one Wisconsin river inorganic mercury levels as high as 684,000 p.p.b. have been noted.

Airborne mercury also contributes to the total human body-burden of mercury. Elevated atmospheric values are noted in the region of mercury mines and refineries. Fernandez et al (1966) observed concentrations of airborne mercury as high as 800,000 ng/m3 close to the Almaden mines in Spain. Mercury vapour has been shown to cross the placental barrier in animals and is taken up by the foetus.

6. Metal induced congenital malformations have been reviewed by Ferm and Hanlon (1983). They conclude that the teratogenic potential of low level chronic exposures to heavy metals has received **little attention** in the past, most current knowledge deriving from examples of acute exposure." It is most probable that the manner in which the human embryo would be exposed to heavy metals is through chronic low-level exposure. Thus, techniques for testing the teratogenic threat of chronic exposure **must be developed"**. (Ferm and Hanlon, op cit). The emphasis is mine. For these reviewers, the possible public health threat of these metals to the human embryo requires further investment by research in heavy metal teratogenesis.

7. According to Settle and Patterson, (1980) the world production of lead has shown a regular increase for almost 5,000 years. Apart from a marked decline coincident with the decline of the Roman Empire the general trend has been upwards, with production accelerating from the time of the first industrial revolution. Its uses in the modern world are legion and it is of particular significance as petrol additives in countries such as the U.K. which have not passed legislation banning its use. I have referred earlier to the implications for the environment globally.

Interest in the effect of lead on health has a quite long history. It is recognised as an environmental hazard in Victorian times and referred to in Roman times. Focussed research interest on the link between environmental lead and children's development is of much more recent origin and a plethora of research reports now in existance span a period of about 15 years; the volume of reported research has increased substantially since the mid 1970s. It is probably the most widely researched of all environmental teratogenic agents, attracting the attention of pediatricians, psychiatrists, neurologists, workers in the field of child health, occupational and industrial medicine,education, various environmental pressure groups and conservationists.Lead has in the past six years been the subject of at least two major national reports. In 1980 the U.K. sponsored Report appeared, titled 'Lead and Health. It represented the considered views of a working party convened by the Department of Health and Social Security under the chairmanship of Professor Lawther with the following terms of reference:

"To review the overall effects on health of envirnmental lead from all sources and, in particular, its effect on the health and development of children, and to assess the contribution lead in petrol makes to the body burden".

In the same year the U.S, National Academy of Sciences published its own report on the subject, and Rutter in England, a member of the Lawther Commitee published a personal paper which reviewed the evidence pertaining to raised lead levels and impaired cognitive and behavioural functioning. In the same year also, Bryce-Smith and Stephens prepared a response to the Lawther Report for the Conservation Society subsequently published as 'Lead or Health'. This was heavily critical of the Working Party's general approach, the nature of evidence reviewed and the weight attached to particular evidence, the method of calculating relative contributions to the total body burden of lead from all sources and the

concessionary ambivalence of final recommendations to what were seen as powerful politico-economic vested interests. The interested reader is referred to these. The bibliographies are instructive. A major disagreement persist, particularly in the U.K., over the relative contribution of air borne lead from vehicle emission, to the lead uptake by pregnant mothers, children and others in the population. There is little disagreement that uptake from the environment is primarily through the gastro-intestinal tract and the lower respiratory tract. Again there is little disagreement that the effects of lead uptake lie on a spectrum which runs through absorption with no detectable change, to absorption with detectable biochemical change of uncertain significance, to poisoning. At one end of the spectrum clinical lead poisoning and its sequelar in children is well documented. Issues of particular significance relate to the possibly more subtle adverse effects on health and development resulting from chronic absorption of lead in smaller quantities than those previously thought to have an observable effect. The effects in question relate to possible impairment of cognitive functioning, behaviour and motor efficiency.

In attempting to balance the evidence on the link between low lead levels and behaviour problems in childhood, the reviewer is faced with results of studies using different methodologies, different methods of attributing and estimating lead burden and different measures of behaviour. The Lawther Committee concluded that having appraised a significent range of representative studies, there was no convincing evidence of deleterious effects of lead at blood levels lower than 35 ug/dl.They had no doubts about neuropsychological consequences of high blood concentration and in particular the evidence of damage and encephalopathy in the region of 80 ugm/dl. Lawther maintained however that effects were doubtful in the region of 35-80 ug/dl.

In this latter context it is interesting to note that Rutter, whilst not dissociating himself from the view of the working party, indicates in his personal review that clinical studies provide good evidence that blood lead levels persistently raised above 60 ug/dl are probably associated with an average reduction of some 3 to 4 IQ points even in children showing no symptoms. He goes further, and suggests that strong pointers exist to the probability of adverse cognitive sequalar in the 40 to 60 ug/dl range, and appears to accept the possibility that impairment may occur at levels within the 20 - 40 ug/dl range. Bryce-Smith and Stephens (1980) go further,, and suggest that the work of David et al (1978), Needleman et al (1979) and Hradina and Winneke (1978) are consistent with the view that impairment of cognitive function and/or behavior may be expected at lead levels as low as 5 ug/dl. It should be noted that under EEC directive (CEC 1977) blood lead surveys must be conducted to determine distribution of values, remedial action being called for if the following protocols are not met:

<20 ug/dl in 50% of group

<30 ug/dl in 90% of group

<35 ug/dl in 98% of group

The U.K. does not meet these in all situation. That a re-examination of lead poisoning levels is necessary appears to be emerging as more recent studies are evaluated. The work of Marlowe and Errera (1982) suggests that lead levels previously thought harmless may be associated with neuro-behavioural impairments. Yule et al (1984) in a replication of Needlemans study (1979) suggest a dose response relationship between difficulties in attention in children and increased blood lead levels. Hyperactivity was also found to be significantly related to children's blood lead levels independant of age. This is consistant with the findings reported by David et al (1976, 1977) which the Lawther Committee effectively dismissed. In a substantial review of behavioural impact of low level lead, Marjorie Smith (1985) concludes that before social factors are controlled "there is an association between higher body levels of lead and poorer performance on IQ tests, even at blood lead levels around 13 ug/dl..... It is also clear that there is in most studies an association between body lead levels and some social factors, although not always social class".This accords well with the view expressed by Bryce-Smith and Stephens (1980) that "no 'no-effect' threshold exists throughtout the whole range of lead burdens now regarded as normal in children, upwards from at least 5 p.p.m. dentine lead". The observations of George Cohen (1980) are salutory:"Our 27 year experience with lead poisoning and the work of others indicate that this is an eradicable disease.... However, so long as there is lead in the environment and children continue to put their hands in their mouths the hazard of poisoning remains". Cohen (op cit) goes on to argue that abatement, public information and screening can all work towards the erradication of risk. The U.S.A. has acted in certain respects in a determined manner. The U.K. has still to do so. An essential first step is the elimination of lead additives from petrol. At a conservative estimate this could directly reduce the blood lead level by between 10 and 20% and indirectly produce greater reduction as the 'fall out' of atmospheric lead diminished with a consequential decrease in lead concentration in urban dust and leaf vegetables.

The effects of maternal lead levels and related foetal lead levels have been less well documented than studies concerned with educational attainment and behaviour of the developing child. Controlled experiments on the foetoxicity of lead are in any case difficult to conduct.Nevertheless as Waldron (1980) notes "What may be of importance in the development of neuropsychiatric abnormalities is the degree to which children are exposed at the time of maximum brain development, which includes exposure in utero". Lead, as Barltrop (1969) pointed out is readily transferred across the human placenta and can be found in foetal tissue. Wibberly et al (1977) reported pronounced mean elevations of lead in placentae from malformed and still births. Bryce-Smith et al (1977) found that the mean lead and cadmium levels in the bones of stillborn/malformed children studied were 5 to 10 times greater than normal. Moreover, Moore, Meredith and Goldberg (1977) showed that early post natal blood lead elevations were significantly associated with an increased risk of mental retardation subsequently. This study and a previous study in Glasgow by Beatie et al 1975 suggests that exposure to lead in utero may be important in the aetiology of mental retardation. A prospective study on the neurological effects of lead in children has been supported by the Greater Glasgow Health Board Research

Group, with an initial population of almost 900 pregnant women (Goldberg 1983). The study is ongoing.

Lead as an environmental teratogen has been recognised for many years. In the U.K. the Health and Safety Executive in draft recommendations published in 1978 propose that pregnant women should be prohibited from work involving exposure to lead and that the maximum blood lead level for women of childbearing age in occupation should be 40 ug/dl. In the U.S.A. the Occupational Safety and Health Administration introduced in 1979 a new standard maximum of 40 ug/dl for **males and females** in recognition of risk to neonates. The OHSA refer to the evidence of risk to infants of prenatal death where either parent has been exposed to lead, and elevated risk for congenital abnormality, retardation and perinatal mortality.

I have spent some time dealing with the effects to children of environmental lead. It is a common element, widely produced, widely used and distributed through the entire ecosphere. In high doses its effects to man have been known for centuries but its low level effects in asymptomatic children are still not fully understood. What is certainly the case is that **its potential for inpairment is greater than was supposed at much lower levels of exposure than are still considered 'safe' by some governments.** This strengthens the argument for compulsory teratological assessment using behavioural criteria for all chemicals introduced into the environment and the rigorous control of pollutants which are known teratogens. Operational concepts such as 'no-effect thresholds' represent at best the overt manifestation of a value system which balances benefit (however defined) against detriment. The complexity of mutagenesis and teratogenesis will mean that to some, the detriment may be substantial and perhaps terminal. Continued lack of knowledge, or precision in relevant knowledge, of effects of chronic low level exposure to environmental hazards is at variance with the aims of a free human society. To be constrained in one's ability to achieve to the full of one's innate potential is to be other than free. To be prevented from functioning normally, either permanently or temporarily as a consequence of a polluted environment is to be assaulted and violated by the actions of others. Action can be taken to prevent the violation:

- rigorous teratological assessment of all new chemicals
- the development of routine screening and monitoring of workers and children for common toxic substances such as lead. This should be routine in pregnancy.
- investment in public health education notably in the case of food additives, and home based hazards eg. painted toys, certain cosmetics from Asia.
- legislation prohibiting the use of organic lead in petrol
- vigorous control of industrial emissions and effluents, particularly heavy metals and known carcinogens. Firmer control of smoke and SO2

- investment by Government in research to explore and develop new techniques and methods for assessing impact of environmental agents on genetic material.

- development of improved quantitative measures for toxic concentration

- the developmentof pro-active control regulations in preference to reaction. Some future problems can be anticipated eg. the environmental build up of drug residues which escape normal methods of sewage control and enter rivers and lakes. Modern society is drug dependant and vast quantities of powerful chemicals enter the environment each year.

- control of foetal health and well being by implementation of research findings in control policy eg. U.S.A. applies blood lead controls to males and females. The UK applies it to females only and as yet these are **recommendations.**

- control of population health and well being by acting on the evidence of evaluated scientific studies of environmental hazard eg. a recent W.H.O. study (Colley and Brasser 1979) in eight European countries investigated the evidence for a relationship between chronic exposure to air pollution and respiratory diseases in children under 11 years. The study evidenced a strong association between smoke pollution and various respiratory indices. The study was **unable to determine a no-effect threshold.** An independent study by Haupt (1984) also reveals a relationship between sedimenting dust and illnesses such as croup. Such studies suggest that control criteria are either inadequate or inadequately enforced.

- a review of the statutory limits for lead in food with a view to reduction.

- a review of therapy studies. Reducing or removing the insulting agent should be explored for benefit accruing. In the same way that dietary adjustment may improve hyperkinesis in some individuals, detoxification may effect amelioration of behavioural difficulties.

The list may be extended. A great need exists for better information on mechanisms, dose response information, for public information and public accountability.

I began this paper with an account of a recent case. I shall end with one.

The concern in recent months over the nuclear reactor fire at Chernobyl has served to heighten international anxiety over safety in the facilities of the nuclear communities; in the UK especially it has exacerbated public alarm regarding radioactive contamination of the environment. A series of incidents at major UK installations had already attracted the continued attention of the press and one such facility - Sellafield in Cumbria - had been subject to scrutiny by the Health and Safety Executive and an all-party Commons inquiry into radio active waste. Amongst other activities, Sellafield is the centre of British Nuclear Fuels reprocessing operation for high level nuclear waste. It acts, in effect, as an international 'nuclear laundry'. There have been nuclear reactors on the site* for almost four decades. 300 nuclear 'incidents' have been recorded in 35 years. (Previously Winscale)

On November 1 1983 Yorkshire Television broadcast a documentary programme which raised serious questions regarding the environmental implications of the Sellafield operation with particular regard to the health and safety of children. The YTV team became interested in cases of childhood cancer during their investigations. Information was collected for the years 1956 - 83 on young people under 22 years old at diagnosis and living in nearby Seascale. Census data provided the necessary information for reaching the conclusion that the incidence of childhood leukaemias in the under 10 years age group represented a **TEN**fold increase when compared with national incidence figures. The YTV team extended their inquiry into the rural district surrounding Sellafield. 25 young people under 22 years old were identified as dying or diagnosed with a cancer condition in the period 1954 - 1983. In the light of these findings and in the absence of any other available explanation the YTV team claimed that the significant high excess of cancer, particularly childhood leukaemia in five parishes south of Sellafield suggested a possible link with environmental radioactivity from Sellafield discharges.

Within three weeks of the YTV broadcast the then Minister of Health had established an independent inquiry under the chairmanship of Sir Douglas Black, and the group had held its first meeting. A Report (Black, 1984) was produced within nine months. The following are among their recommendations and conclusions.

"The hypothesis ..... that the proximity of Sellafield to the village of Seascale could be a factor in producing cases of childhood leukaemia is not one which can be categorically dismissed, nor, on the other hand is it easy to prove" (6.2).

"We have **found no evidence of any general risk to health** for children or adults living near Sellafield when compared to the rest of Cumbria and **we can give a qualified reassurance to the people** who are concerned

about a possible health hazard in the neighbourhgood of Sellafield". (6,13).

"It is **impossible to establish for certain** the situation with regard to environmental levels of radiation around Sellafield twenty or thirty years ago, and **we shall never know the actual doses received** by these children subsequently contracting leukaemia". (6,10). (Authors italics)

The Black Committee accepted that significant doses to humans could be delivered by unplanned discharges of radioactive waste. The committee **accepted** the National Radiological Protection Board's 'best estimate' of the average radiation dose to the **red bone marrow** received by a model population of young people in Seascale. **They were surprised at the few data on body levels of radionuclides** in local people not employed at the plant. **They were surprised** at the **lack of health co-ordination in the assessment of discharges impact** on the population. The Committee paid particular attention to comparing the doses received from background to those from the Sellafield discharges. Evidence was received from **whole body monitoring** and agree that "it is much more difficult to detect the alpha emitters, plutonium and americium in this way, and we were **unable to obtain any data on alpha emitter levels in children**". (4,66).

Now, it has been known for decades that ionising radiation can produce genetic changes; it is essentially a mutagenic agent and as such may act upon the germinal or somatic cells of the genetic apparatus. Sharratt (op cit) has summarised the possibilities, amongst which is the birth of a child with genetically induced disease or predisposition to disease. Renwick (op cit) essentially agrees, and notes that ionising radiation can mutate the DNA in the germ-line or it can kill sensitive cells in the embryo in utero if applied at the right time. In this sense ionising radiation can act as a mutagen and teratogen. It has been previously emphasised in this paper that for a known teratogen **there is no safe dosage level.**

Over the time span of the YTV enquiry substantial radiation leaks have occurred from the Sellafield site. Between 1952 - 1955 20 kilogrammes of uranium were discharged into the atmospehre. In 1957 fire destroyed the core of a plutonium producing reactor resulting in rigorous control of local produce, especially dairy produce. Some later reports drew a link between the environmental contamination and upwards of 260 cases of cancer. Officially the link remained unconfirmed. Cases of worker contamination occurred at intervals through the 70s and in November 1983 a substantial unregulated discharge of radioactive waste closed the beaches within 25 miles of Sellafield. Periodic unregulated discharge of radioactive material is an aspect of the history of the plant. Periodic regulated discharges of low level radioactive waste is part of its normal operating procedure. It must be borne in mind that the operation of a nuclear reprocessing plant creates vast problems of storage. The Friends of the Earth have calculated that reprocessing increases the volume of waste by a factor of 160. The Guardian (26 February 1986) reports that one forecast by the Department of the Environment puts the storage problem for low level waste in the nuclear industry at half a million cubic metres. On Wednesday 12 March 1986, an all party Commons Committee condemned the U.K. approach to nuclear waste

disposal as amateurish, haphazard and complacent. At the dumping site near Sellafield rain water is allowed to drain through earth trenches containing unpackaged, unlabelled waste and ultimately finds its way to the Irish sea. (Guardian 13 March 1986).

Reports in the Sunday Times (February 23 1986) further undermine confidence in the industry and credibility of the Black Committee's findings. According to this Report evidence given to the Black Committee by the nuclear industry was grossly misleading and doses of radiation received by the local population in the early 1950s were five times higher than the figure given to the Committee.

There is a further point which undermines the credibility of the Black Report. The NRPB provided evidence of nuclear take-up by children as evidenced in studies of red bone marrow. No other sites were investigated, particularly the doses to lymph nodes and lymphoid tissue. Equally the most dangerous sources of radiation to the foetus, ie. plutonium and americium were beyond the scope of NRPBs data gathering. Some estimates (Guardian, 8 April 1986) put the risk to the foetus from aplha emitters like Plutonium and Americium, as 200 times greater than the risk of damage from a similar dose of X-rays.

The official position still is however that the link between Sellafield discharges and childhood cancers is not proven.

On December 3 1985 YTV followed up its award winning documentary with the first ever detailed televison investigation of Britain's nuclear weapons industry. In the complete absence of official environmental studies of impact on health in communities around bomb factories and nuclear defence installations the YTV team carried out their own enquiry. At Aldermaston, the main bomb factory there is a history of accidents and contamination. At nuclear submarine bases in Scotland, both U.S.N. and R.N., the beaches and mudbanks show evidence of radioactive contamination. The pattern of child health in all the sites examined show incidences of childhood leukaemia and lymphatic cancers which vary between 3 and 10 times the national average. Can these all be co-incidences? The fact that local hot-spots can show higher than average incidences of cancer-related diseases for inexplicable reasons has to be taken alongside the **consistent demonstration of higher than average risks** close to facilities where radio-active material is fabricated, stored, used - and sometimes ineffectively regulated. Can the violation of childhood continue amidst an 'official' stance which is both reluctant to initiate properly controlled health monitoring studies, or countenance the validity of a causal hypothesis.

Whether we are concerned for the effects of dioxins, radio elements or metallic neurotoxins, the vulnerable stages of infancy and childhood have a right to the highest regard the community can pay. Part of that price is the greater public accountability of 'national' and private industrial enterprises.

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